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Main Menu | Search Form | Posting Counts | Show S Numbers | Edit S Numbers | Preferences | Cases

Search Results -

Term	Documents
"REPORTER CONSTRUCT".USPT.	0
@PD.USPT.	7064522
((4 AND "REPORTER CONSTRUCT") AND ((@PD > "20020916")!)).USPT.	2
((L4 AND "REPORTER CONSTRUCT") AND ((@PD > 20020916)!)).USPT.	2

Database: US Patents Full-Text Database; US Pre-Grant Publication Full-Text Database; JPO Abstracts Database; EPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Search: L5

Refine Search | Recall Text | Clear

Search History

DATE: Friday, April 25, 2003 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
	DB=USPT; THES=ASSIGNEE; PLUR=YES; OP=OR		
L5	(L4 and "reporter construct") AnD ((@pd > 20020916)!)	2	L5
L4	(L3 and (screen or assay)) AnD ((@pd > 20020916)!)	4	L4
L3	(L2 and reporter) AnD ((@pd > 20020916)!)	4	L3
L2	(L1 and apoptosis) AnD ((@pd > 20020916)!)	12	L2
L1	(p300 or cbp) AnD ((@pd > 20020916)!)	79	L1

END OF SEARCH HISTORY

NPL search 09/674,876

Your SELECT statement is:
s (p300 or cbp) and mdm2

Items	File
26	5: Biosis Previews(R)_1969-2002/Sep W1
42	34: SciSearch(R) Cited Ref Sci_1990-2002/Sep W3
4	35: Dissertation Abs Online_1861-2002/Aug
1	65: Inside Conferences_1993-2002/Sep W3
24	71: ELSEVIER BIOBASE_1994-2002/Sep W2
28	73: EMBASE_1974-2002/Sep W2
1	94: JICST-EPlus_1985-2002/Jul W3
4	98: General Sci Abs/Full-Text_1984-2002/Aug
3	144: Pascal_1973-2002/Sep W3
2	149: TGG Health&Wellness DB(SM)_1976-2002/Sep W2
35	155: MEDLINE(R)_1966-2002/Sep W2
4	156: ToxFile_1965-2002/Sep W2
32	159: Cancerlit_1975-2002/Aug
5	172: EMBASE Alert_2002/Sep W3
5	266: FEDRIP_2002/Jul
1	370: Science_1996-1999/Jul W3
25	399: CA SEARCH(R)_1967-2002/UD=13712

SYSTEM:OS - DIALOG OneSearch
File 34:SciSearch(R) Cited Ref Sci 1990-2002/Sep W3
(c) 2002 Inst for Sci Info
***File 34: Alert feature enhanced for multiple files, duplicates** removal, customized scheduling. See HELP ALERT.
File 73:EMBASE 1974-2002/Sep W2
(c) 2002 Elsevier Science B.V.
***File 73: Alert feature enhanced for multiple files, duplicates** removal, customized scheduling. See HELP ALERT.
File 155:MEDLINE(R) 1966-2002/Sep W2
***File 155: Alert feature enhanced for multiple files, duplicates** removal, customized scheduling. See HELP ALERT.
File 159:Cancerlit 1975-2002/Aug
(c) format only 2002 Dialog Corporation

Set	Items	Description
S1	137	(CBP OR P300) AND MDM2
S2	13	S1 NOT PY=>1998
S3	6	RD (unique items)

3/9/2 (Item 2 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2002 Inst for Sci Info. All rts. reserv.

05885870 Genuine Article#: XF144 Number of References: 30
Title: Synergistic activation of transcription by CBP **and p53**
Author(s): Gu W; Shi XL; Roeder RG (REPRINT)
Corporate Source: ROCKEFELLER UNIV,BIOCHEM & MOL BIOL LAB, 1230 YORK AVE/NEW YORK//NY/10021 (REPRINT); ROCKEFELLER UNIV,BIOCHEM & MOL BIOL LAB/NEW YORK//NY/10021
Journal: NATURE, 1997, V387, N6635 (JUN 19), P819-823
ISSN: 0028-0836 Publication date: 19970619
Publisher: MACMILLAN MAGAZINES LTD, PORTERS SOUTH, 4 CRINAN ST, LONDON, ENGLAND N1 9XW
Language: English Document Type: ARTICLE
Geographic Location: USA
Subfile: CC PHYS--Current Contents, Physical, Chemical & Earth Sciences; CC LIFE--Current Contents, Life Sciences; CC AGRI--Current Contents, Agriculture, Biology & Environmental Sciences;
Journal Subject Category: MULTIDISCIPLINARY SCIENCES
Abstract: The tumour suppressor p53 is a transcriptional regulator whose ability to inhibit cell growth is dependent upon its transactivation function(1-3). Here we demonstrate that the transcription factor **CBP**, which is also implicated in cell proliferation and differentiation(4-14), acts as a p53 coactivator and potentiates its transcriptional activity. The amino-terminal activation domain of p53 interacts with the carboxy-terminal portion of the **CBP** protein both

in vitro and in vivo. In transfected SaoS-2 cells, **CBP** potentiates activation of the mdm-2 gene by p53 and, reciprocally, p53 potentiates activation of a Gal4-responsive target gene by a Gal4(1-147)- **CBP** (1678-2441) fusion protein. A double point mutation that destroys the transactivation function of p53 also abolishes its binding to **CBP** and its synergistic function with **CBP** The ability of p53 to interact physically and functionally with a coactivator (**CBP**) that has histone acetyltransferase activity(15,16) and with components (TAFs)(17,18) of the general transcription machinery indicates that it may have different functions in a multistep activation pathway.

3/9/3 (Item 3 from file: 34)

DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05794560 Genuine Article#: WY183 Number of References: 79

Title: Immortalization of primary epithelial cells by E1A 12S requires late, second exon-encoded functions in addition to complex formation with pRB and p300

Author(s): Gopalakrishnan S; Douglas JL; Quinlan MP (REPRINT)
Corporate Source: UNIV TENNESSEE,CTR HLTH SCI, DEPT MICROBIOL & IMMUNOL, 858 MADISON AVE/MEMPHIS//TN/38163 (REPRINT); UNIV TENNESSEE,CTR HLTH SCI, DEPT MICROBIOL & IMMUNOL/MEMPHIS//TN/38163

Journal: CELL GROWTH & DIFFERENTIATION, 1997, V8, N5 (MAY), P541-551
ISSN: 1044-9523 Publication date: 19970500
Publisher: AMER ASSOC CANCER RESEARCH, PUBLIC LEDGER BLDG, SUITE 816, 150 S. INDEPENDENCE MALL W., PHILADELPHIA, PA 19106
Language: English Document Type: ARTICLE
Geographic Location: USA
Subfile: CC LIFE--Current Contents, Life Sciences
Journal Subject Category: BIOCHEMISTRY & MOLECULAR BIOLOGY; CELL BIOLOGY

Abstract: Immortalization of primary cells is an early and important event in multistep tumorigenesis and is itself a multistep process, Adenovirus E1A 12S encodes an oncoprotein that can rescue cells from senescence and overcome apoptosis, leading to their immortalization, Five regions of 12S, located in both exons, are required for immortalization, Two regions in the first exon are necessary to activate the cell cycle, increase the number of population doublings, and overcome the M1 stage of mortality, However, extension of life span requires overcoming crisis or M2, which can be accomplished by the expression of the second exon, Several cellular proteins associate with the peptide encoded by the first exon of 12S including pRB, p107, p130, and **p300** . The importance of pRB-E1A and **p300** -E1A complexes in transformation is well established; however, their roles in 12S-mediated immortalization remain undefined, Results obtained from the present study using a panel of second exon immortalization-defective mutants demonstrate that formation of pRB-E1A and **p300** -E1A complexes is insufficient for immortalization of primary cells, We further demonstrate that the expression levels of another tumor suppressor protein, p53, also do not correlate with the inability of the mutants to immortalize, Thus, mutations in the second exon of 12S do not affect the early steps in the immortalization pathway, The second exon mutants are defective in performing a late function in immortalization, involving the reactivation of the cell cycle, indicating that it is a crucial event in immortalization.

3/9/4 (Item 4 from file: 34)

DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05631420 Genuine Article#: WM070 Number of References: 54

Title: Inhibition of p53-mediated transactivation and cell cycle arrest by E1A through its p300 **/ CBP -interacting region**

Author(s): Somasundaram K; ElDeiry WS (REPRINT)
Corporate Source: UNIV PENN,SCH MED, DEPT MED & GENET, LAB MOL ONCOL & CELL CYCLE REGUL, HOWARD HUGHES /PHILADELPHIA//PA/19104 (REPRINT); UNIV PENN,SCH MED, DEPT MED & GENET, LAB MOL ONCOL & CELL CYCLE REGUL, HOWARD HUGHES /PHILADELPHIA//PA/19104; CTR COMPREHENS CANC,/PHILADELPHIA//PA/19104

Journal: ONCOGENE, 1997, V14, N9 (MAR 6), P1047-1057
ISSN: 0950-9232 Publication date: 19970306

Publisher: STOCKTON PRESS, HOUNDMILLS, BASINGSTOKE, HAMPSHIRE, ENGLAND RG21 6XS
Language: English Document Type: ARTICLE
Geographic Location: USA
Subfile: CC LIFE--Current Contents, Life Sciences
Journal Subject Category: ONCOLOGY; BIOCHEMISTRY & MOLECULAR BIOLOGY; CELL BIOLOGY
Abstract: Cellular transformation by the adenovirus E1A oncoprotein requires its **p300 / CBP** - and Rb-binding domains. We mapped inhibition of p53-mediated transactivation to the **p300 / CBP** -binding region of E1A. An E1A mutant incapable of physically interacting with Rb retained the capacity to inhibit transactivation by p53, whereas E1A mutants of the **p300 / CBP** -interacting domain failed to inhibit p53. The inhibitory effect of the **p300 / CBP** -binding region of E1A on p53 was demonstrated with p53-activated reporters and endogenous p53 targets such as p21(WAF1/CIP1) or **MDM2** . E1A lacking the capacity to interact with Rb, but capable of **p300 / CBP** interaction, was competent in suppression of a DNA-damage activated p53-dependent cell cycle checkpoint. Exogenous **CBP** and **p300** were able to individually relieve EIA's inhibitory effect on p53-mediated transcription. Mutants of E1A that are not capable of interacting with **p300** or **CBP** were found to efficiently stabilize endogenous p53 but were not competent in repression of p21 expression thus dissociating these two effects of E1A. Our results suggest that the **p300 / CBP** -binding domain of E1A inhibits a p53-dependent cellular response which normally inhibits DNA replication following Adenovirus infection.

3/9/5 (Item 5 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05356182 Genuine Article#: VT249 Number of References: 36
Title: THE CBP **COACTIVATOR STIMULATES E2F1/DP1 ACTIVITY**
Author(s): TROUCHE D; COOK A; KOUZARIDES T
Corporate Source: WELLCOME CRC INST,TENNIS COURT RD/CAMBRIDGE CB21QR//ENGLAND/; WELLCOME CRC INST/CAMBRIDGE CB2 1QR//ENGLAND/; UNIV CAMBRIDGE,DEPT PATHOL/CAMBRIDGE CB2 1QR//ENGLAND/
Journal: NUCLEIC ACIDS RESEARCH, 1996, V24, N21 (NOV 1), P4139-4145
ISSN: 0305-1048
Language: ENGLISH Document Type: ARTICLE
Geographic Location: ENGLAND
Subfile: SciSearch; CC LIFE--Current Contents, Life Sciences
Journal Subject Category: BIOCHEMISTRY & MOLECULAR BIOLOGY
Abstract: The cell cycle-regulating transcription factors E2F1/DP1 activate genes whose products are required for S phase progression. During most of the G1 phase, E2F1/DP1 activity is repressed by the retinoblastoma gene product RB, which directly contacts the E2F1 activation domain and silences it. The E2F1 activation domain has sequence similarity to the N-terminal activation domain of E1A(12S), which contains binding sites for **CBP** as well as RB. Here, we present evidence that the **CBP** protein directly contacts E2F1/DP1 and stimulates its activation capacity. We show that **CBP** interacts with the activation domain of E2F1 both in vitro and in vivo. Deletion of four residues from the E2F1 activation domain reduces **CBP** binding as well as transcriptional activation, but still allows the binding of RB and **MDM2** . This deletion removes residues which are conserved in the N-terminal activation domain of E1A and which are required for the binding of **CBP** to E1A. When the E1A N-terminus is used as a competitor in squelshing experiments it abolishes **CBP** -induced activation of E2F1/DP1, whereas an E1A mutant lacking **CBP** binding ability fails to do so. These results indicate that **CBP** can act as a coactivator for E2F1 and suggest that **CBP** recognises a similar motif within the E1A and E2F1 activation domains. The convergence of the RB and **CBP** pathways on the regulation of E2F1 activity may explain the cooperativity displayed by these proteins in mediating the biological functions of E1A. We propose a model in which E1A activates E2F not only by removing the RB repression but also by providing the **CBP** co-activator.

SYSTEM:OS - DIALOG OneSearch
File 4:INSPEC 1983-2002/Sep W3
(c) 2002 Institution of Electrical Engineers
***File 4: Alert feature enhanced for multiple files, duplicates** removal, customized scheduling. See HELP ALERT.
File 155:MEDLINE(R) 1966-2002/Sep W3
***File 155: Alert feature enhanced for multiple files, duplicates** removal, customized scheduling. See HELP ALERT.
File 159:Cancerlit 1975-2002/Aug
(c) format only 2002 Dialog Corporation

Set	Items	Description
?s p300 and stable(w)transfect?		
	4044	P300
	288964	STABLE
	143001	TRANSFECT?
	3197	STABLE(W)TRANSFECT?
S1	5	P300 AND STABLE(W)TRANSFECT?
?rd		
...completed examining records		
S2	3	RD (unique items)

?t/full/1

2/9/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12750062 21617170 PMID: 11741590
Modulation of HIV-1 enhancer activity and virus production by cAMP.
Banas B; Eberle J; Banas B; Schlondorff D; Luckow B
Medizinische Poliklinik, Ludwig-Maximilians-Universitat Munchen, Molekulare Infektiologie, Pettenkoferstrasse 8a, D-80336 Munich, Germany. banas@medpoli.med.uni-muenchen.de
FEBS letters (Netherlands) Dec 7 2001, 509 (2) p207-12, ISSN 0014-5793 Journal Code: 0155157
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS
The effect of cAMP on the transcriptional activity of the HIV-1 long terminal repeat/enhancer was investigated and compared to the effect of cAMP on virus replication. In culture cAMP repressed virus replication in vivo using different cell types. Transient transfection studies with HIV-1 enhancer-derived luciferase reporter gene constructs identified the minimal DNA sequence mediating the negative regulatory effect of cAMP on HIV-1 transcription. A single nuclear factor kappaB element from the HIV-1 enhancer mediates the repressive effect on transcription. AP-2 is not involved in cAMP repression. **Stable transfection** of Jurkat T cells with the co-activators CREB binding protein (CBP) and **p300** completely abolished the cAMP repressive effect, supporting the hypothesis that elevation of intracellular cAMP increases phosphorylation of CREB, which then competes with phosphorylated p65 and Ets-1 for limiting amounts of CBP/ **p300** thereby mediating the observed repressive effect on transcription. These findings suggest an important role of cAMP on HIV-1 transcription.
Tags: Support, Non-U.S. Gov't
Descriptors: *Cyclic AMP--pharmacology--PD; *HIV Enhancer--drug effects --DE; *HIV-1--drug effects--DE; *Lymphocytes--virology--VI; *Virus Replication--drug effects--DE; Binding Sites; Cell Line; DNA-Binding Proteins--metabolism--ME; HIV-1--genetics--GE; NF-kappa B--metabolism--ME; Nuclear Proteins--metabolism--ME; Nucleic Acid Synthesis Inhibitors --pharmacology--PD; Trans-Activators--metabolism--ME; Transcription Factors --metabolism--ME; Transcription, Genetic--drug effects--DE
CAS Registry No.: 0 (CREB-binding protein); 0 (DNA-Binding Proteins); 0 (E1A-associated p300 protein); 0 (NF-kappa B); 0 (Nuclear Proteins) ; 0 (Nucleic Acid Synthesis Inhibitors); 0 (Trans-Activators); 0 (Transcription Factors); 0 (enhancer-binding protein AP-2); 60-92-4 (Cyclic AMP)

Record Date Created: 20011219
?t/full/2

2/9/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10409418 99410419 PMID: 10480893
Requirement of an E1A-sensitive coactivator for long-range transactivation by the beta-globin locus control region.
Forsberg E C; Johnson K; Zaboikina T N; Mosser E A; Bresnick E H
Department of Pharmacology, University of Wisconsin Medical School, Madison, Wisconsin 53706, USA.
Journal of biological chemistry (UNITED STATES) Sep 17 1999, 274 (38) p26850-9, ISSN 0021-9258 Journal Code: 2985121R
Contract/Grant No.: DK50107; DK; NIDDK
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS
Four erythroid-specific DNase I-hypersensitive sites at the 5'-end of the beta-globin locus confer high-level transcription to the beta-globin genes. To identify coactivators that mediate long-range transactivation by this locus control region (LCR), we assessed the influence of E1A, an inhibitor of the CBP/ **p300** histone acetylase, on LCR function. E1A strongly inhibited transactivation of Agamma- and beta-globin promoters by the HS2, HS2-HS3, and HS1-HS4 subregions of the LCR in human K562 and mouse erythroleukemia cells. Short- and long-range transactivation mediated by the LCR were equally sensitive to E1A. The E1A sensitivity was apparent in transient and **stable transfection** assays, and E1A inhibited expression of the endogenous gamma-globin genes. Only sites for NF-E2 within HS2 were required for E1A sensitivity in K562 cells, and E1A abolished transactivation mediated by the activation domain of NF-E2. E1A mutants defective in CBP/ **p300** binding only weakly inhibited HS2-mediated transactivation, whereas a mutant defective in retinoblastoma protein binding strongly inhibited transactivation. Expression of CBP/ **p300** potentiated HS2-mediated transactivation. Moreover, expression of GAL4-CBP strongly increased transactivation of a reporter containing HS2 with a GAL4 site substituted for the NF-E2 sites. Thus, we propose that a CBP/ **p300** -containing coactivator complex is the E1A-sensitive factor important for LCR function.
Tags: Animal; Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
Descriptors: *Adenovirus E1A Proteins--metabolism--ME; *Adenovirus E1A Proteins--pharmacology--PD; *Globins--genetics--GE; *Locus Control Region; *Nuclear Proteins--antagonists and inhibitors--AI; *Trans-Activation (Genetics); *Trans-Activators--antagonists and inhibitors--AI; Acetyltransferases--metabolism--ME; DNA-Binding Proteins--metabolism--ME; Leukemia, Erythroblastic, Acute--genetics--GE; Leukemia, Erythroblastic, Acute--metabolism--ME; Mice; Polymerase Chain Reaction; Transcription Factors--metabolism--ME; Tumor Cells, Cultured
CAS Registry No.: 0 (Adenovirus E1A Proteins); 0 (DNA-Binding Proteins); 0 (E1A-associated p300 protein); 0 (Nuclear Proteins); 0 (Trans-Activators); 0 (Transcription Factors); 125267-48-3 (erythroid-specific DNA-binding factor); 9004-22-2 (Globins)
Enzyme No.: EC 2.3.1. (Acetyltransferases); EC 2.3.1.48 (histone acetyltransferase)
Record Date Created: 19991013
?t/full/3

2/9/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10226930 99223576 PMID: 10207071
Cooperation between phosphorylation and acetylation processes in transcriptional control.
Espinos E; Le Van Thai A; Pomies C; Weber M J
Laboratoire de Biologie Moleculaire Eucaryote, CNRS UPR 9006, 31062 Toulouse Cedex, France.
Molecular and cellular biology (UNITED STATES) May 1999, 19 (5)

p3474-84, ISSN 0270-7306 Journal Code: 8109087
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS

We previously reported that the activation of the M promoter of the human choline acetyltransferase (ChAT) gene by butyrate and trapoxin in transfected CHP126 cells is blocked by PD98059, a specific mitogen-activated protein kinase kinase (MEK) inhibitor (E. Espinos and M. J. Weber, Mol. Brain Res. 56:118-124, 1998). We now report that the transcriptional effects of histone deacetylase inhibitors are mediated by an H7-sensitive serine/threonine protein kinase. Activation of the ChAT promoter by butyrate and trapoxin was blocked by 50 microM H7 in both transient- and **stable - transfection** assays. Overexpression of **p300** , a coactivator protein endowed with histone acetyltransferase activity, stimulated the ChAT promoter and had a synergistic effect on butyrate treatment. These effects were blocked by H7 and by overexpressed adenovirus E1A 12S protein. Moreover, both H7 and PD98059 suppressed the activation of the Rous sarcoma virus (RSV) and simian virus 40 promoters by butyrate in transfection experiments. Similarly, the induction of the cellular histone H1(0) gene by butyrate in CHP126 cells was blocked by H7 and by PD98059. Previous data (L. Cuisset, L. Tichonicky, P. Jaffray, and M. Delpech, J. Biol. Chem. 272:24148-24153, 1997) showed that the induction of the H1(0) gene by butyrate is blocked by okadaic acid, an inhibitor of protein phosphatases. We now show that the activation of the ChAT and RSV promoters by butyrate in transfected CHP126 cells is also blocked by 200 nM okadaic acid. Western blotting and in vivo metabolic labeling experiments showed that butyrate has a biphasic effect on histone H3 phosphorylation, i.e., depression for up to 16 h followed by stimulation. The data thus strongly suggest that the transcriptional effects of histone deacetylase inhibitors are mediated through the activation of MEK1 and of an H7-sensitive protein kinase in addition to protein phosphatases.

Tags: Human; Support, Non-U.S. Gov't

Descriptors: *Gene Expression Regulation--genetics--GE; *Protein-Serine-Threonine Kinases--genetics--GE; 1-(5-Isoquinolinesulfonyl)-2-methylpiperazine--pharmacology--PD; Acetylation; Acetyltransferases--genetics--GE; Adenovirus E1A Proteins--genetics--GE; Antibiotics, Peptide--pharmacology--PD; Butyrates--pharmacology--PD; Cell Cycle--genetics--GE; Cell Cycle Proteins--genetics--GE; Cell Line; Choline O-Acetyltransferase--genetics--GE; Enzyme Inhibitors--pharmacology--PD; Flavones--pharmacology--PD; Genes, Reporter--genetics--GE; Histone Deacetylase --antagonists and inhibitors--AI; Histones--genetics--GE; Okadaic Acid--pharmacology--PD; Phosphorylation; Promoter Regions (Genetics)--genetics--GE; Trans-Activation (Genetics)--drug effects--DE; Transfection

CAS Registry No.: 0 (Adenovirus E1A Proteins); 0 (Antibiotics, Peptide); 0 (Butyrates); 0 (Cell Cycle Proteins); 0 (Enzyme Inhibitors); 0 (Flavones); 0 (Histones); 0 (PD 98059); 0 (p300-CBP-associated factor); 133155-89-2 (trapoxin A); 78111-17-8 (Okadaic Acid); 84477-87-2 (1-(5-Isoquinolinesulfonyl)-2-methylpiperazine)

Enzyme No.: EC 2.3.1. (Acetyltransferases); EC 2.3.1.6 (Choline O-Acetyltransferase); EC 2.7.1.- (Protein-Serine-Threonine Kinases); EC 3.5.1.- (Histone Deacetylase)

Record Date Created: 19990518

?ds

Set	Items	Description
S1	5	P300 AND STABLE(W)TRANSFECT?
S2	3	RD (unique items)

09/674,876

Your SELECT statement is:
s (cbp or p300) and apoptosis

Items	File
123	5: Biosis Previews(R)_1969-2002/Sep W1
155	34: SciSearch(R) Cited Ref Sci_1990-2002/Sep W3
12	35: Dissertation Abs Online_1861-2002/Aug
1	65: Inside Conferences_1993-2002/Sep W3
89	71: ELSEVIER BIOBASE_1994-2002/Sep W2
103	73: EMBASE_1974-2002/Sep W2
1	77: Conference Papers Index_1973-2002/Sep
8	94: JICST-EPlus_1985-2002/Jul W3
23	98: General Sci Abs/Full-Text_1984-2002/Aug
2	135: NewsRx Weekly Reports_1995-2002/Sep W2
27	144: Pascal_1973-2002/Sep W3
11	149: TGG Health&Wellness DB(SM)_1976-2002/Sep W2
116	155: MEDLINE(R)_1966-2002/Sep W2
17	156: ToxFile_1965-2002/Sep W2
98	159: Cancerlit_1975-2002/Aug
1	162: CAB Health_1983-2002/Aug
12	172: EMBASE Alert_2002/Sep W3
32	266: FEDRIP_2002/Jul
2	370: Science_1996-1999/Jul W3
64	399: CA SEARCH(R)_1967-2002/UD=13712
3	442: AMA Journals_1982-2002/Aug B1
1	444: New England Journal of Med._1985-2002/Sep W3

SYSTEM:OS - DIALOG OneSearch
File 5:Biosis Previews(R) 1969-2002/Sep W1
(c) 2002 BIOSIS
***File 5: Alert feature enhanced for multiple files, duplicates** removal, customized scheduling. See HELP ALERT.
File 34:SciSearch(R) Cited Ref Sci 1990-2002/Sep W3
(c) 2002 Inst for Sci Info
***File 34: Alert feature enhanced for multiple files, duplicates** removal, customized scheduling. See HELP ALERT.
File 73:EMBASE 1974-2002/Sep W2
(c) 2002 Elsevier Science B.V.
***File 73: Alert feature enhanced for multiple files, duplicates** removal, customized scheduling. See HELP ALERT.
File 155:MEDLINE(R) 1966-2002/Sep W2
***File 155: Alert feature enhanced for multiple files, duplicates** removal, customized scheduling. See HELP ALERT.
File 159:Cancerlit 1975-2002/Aug
(c) format only 2002 Dialog Corporation

Set	Items	Description
S1	595	(CBP OR P300) AND APOPTOSIS
S2	144	S1 AND PROMOTER?
S3	29	S2 NOT PY=>1998
S4	8	RD (unique items)

4/9/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2002 BIOSIS. All rts. reserv.

11018112 BIOSIS NO.: 199799639257
Recruitment of p300 / CBP **in p53-dependent signal pathways.**
AUTHOR: Avantaggiati Maria Laura; Ogryzko Vasily; Gardner Kevin; Giordano Antonio; Levine Arthur S; Kelly Kathleen(a)
AUTHOR ADDRESS: (a)Lab. Pathol., Natl. Cancer Inst., Natl. Inst. Health, Bethesda, MD 20892**USA
JOURNAL: Cell 89 (7):p1175-1184 1997
ISSN: 0092-8674
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The products of the p53 and **CBP / p300** genes have been individually implicated in control of cell growth and regulation of transcription. p53 is known to act as a positive and negative regulator of gene expression. Here we show that p53, in both wild-type and mutant

conformation, forms a specific protein complex with **p300** . However, in its wild-type but not mutant conformation, p53 inhibits a **promoter** containing the DNA-binding sequences for the transcription factor AP1, in a **p300** -dependent manner. **p300** stimulates the transcriptional activity of p53 on p53-regulated **promoters** , and it enhances the responsiveness to a physiological upstream modulator of p53 function, ionizing radiation. A dominant negative form of **p300** prevents transcriptional activation by p53, and it counteracts p53-mediated G1 arrest and **apoptosis** . The data implicate **p300** as an important component of p53-signaling, thus providing new insight into the mechanisms of cellular proliferation.

4/9/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

10985947 BIOSIS NO.: 199799607092
Binding and modulation of p53 by p300 / CBP **coactivators.**
AUTHOR: Lill Nancy L; Grossman Steven R; Ginsberg Doron; Decaprio James; Livingston David M(a)
AUTHOR ADDRESS: (a)Dana-Farber Cancer Inst., Harvard Med. Sch., 44 Binney St., Boston, MA 02115**USA
JOURNAL: Nature (London) 387 (6635):p823-827 1997
ISSN: 0028-0836
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The adenovirus E1A and SV40 large-T-antigen oncoproteins bind to members of the **p300 / CBP** transcriptional coactivator family. Binding of **p300 / CBP** is implicated in the transforming mechanisms of E1A and T-antigen oncoproteins. A common region of the T antigen is critical for binding both **p300 / CBP** and the tumour suppressor p53 (ref. 1), suggesting a link between the functions of p53 and **p300** . Here we report that **p300 / CBP** binds to p53 in the absence of viral oncoproteins, and that **p300** and p53 colocalize within the nucleus and coexist in a stable DNA-binding complex. Consistent with its ability to bind to **p300** , E1A disrupted functions mediated by p53. it reduced p53-mediated activation of the p21 and bax **promoters** , and suppressed p53-induced cell-cycle arrest and **apoptosis** . We conclude that members of the **p300 / CBP** family are transcriptional adaptors for p53, modulating its checkpoint function in the G1 phase of the cell cycle and its induction of **apoptosis** . Disruption of **p300** /p53-dependent growth control may be part of the mechanism by which E1A induces cell transformation. These results help to explain how p53 mediates growth and checkpoint control, and how members of the **p300 / CBP** family affect progression from G1 to the S phase of the cell cycle.

4/9/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

10898776 BIOSIS NO.: 199799519921
Accumulation of p53 induced by the adenovirus E1A protein requires regions involved in the stimulation of DNA synthesis.
AUTHOR: Querido Emmanuelle; Teodoro Jose G; Branton Philip E(a)
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JOURNAL: Journal of Virology 71 (5):p3526-3533 1997
ISSN: 0022-538X
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: It has been known for some time that expression of the 243-residue (243R) human adenovirus type 5 (Ad5) early region 1A (E1A) protein causes an increase in the level of the cellular tumor suppressor p53 and induction of p53-dependent **apoptosis** . Deletion of a portion of conserved region 1 (CR1) had been shown to prevent **apoptosis** , suggesting that binding of **p300** and/or the pRB retinoblastoma tumor suppressor and related proteins might be implicated. To examine the

mechanism of the E1A-induced accumulation of p53, cells were infected with viruses expressing E1A-243R containing various deletions which have well-characterized effects on **p300** and pRB binding. It was found that in human HeLa cells and rodent cells, complex formation with **p300** but not pRB was required for the rise in p53 levels. However, in other human cell lines, including MRC-5 cells, E1A proteins which were able to form complexes with either **p300** or pRB induced a significant increase in p53 levels. Only E1A mutants defective in binding both classes of proteins were unable to stimulate p53 accumulation. This same pattern was also apparent in p53-null mouse cells coinfected by Ad5 mutants and an adenovirus vector expressing either wild-type or mutant human p53 under a cytomegalovirus **promoter** , indicating that the difference in importance of pRB binding may relate to differences between rodent and human p53 expression. The increase in p53 levels correlated well with the induction of **apoptosis** and, as shown previously, with the stimulation of cellular DNA synthesis. Thus, it is possible that the accumulation of p53 is induced by the induction of unscheduled DNA synthesis by E1A proteins and that increased levels of p53 then activate cell death pathways.

4/9/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

10669183 BIOSIS NO.: 199799290328
Human E2F-1 reactivates cell cycle progression in ventricular myocytes and represses cardiac gene transcription.
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JOURNAL: Developmental Biology 179 (2):p402-411 1996
ISSN: 0012-1606
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The "pocket" protein- and **p300** -binding domains of E1A mediate alternative pathways that, independently, provoke S phase reentry in ventricular muscle cells and repress cardiac-specific transcription. In the present study, we utilized recombinant adenovirus to deliver mammalian E2F-1, whose release from pocket proteins may underlie effects of E1A and mitogenic signaling. Like E1A, E2F-1 proved cytotoxic in the absence of E1B. Used along with E1B to avert **apoptosis** , E2F-1 inhibited the cardiac and skeletal a-actin **promoters** , serum response factor abundance, and sarcomeric actin biosynthesis, while inducing DNA synthesis and proliferating cell nuclear antigen. Image analysis of Feulgen-stained nuclei corroborated a parallel increase in DNA content, with accumulation in G-2/M. Thus, E2F-1 suffices for all observed actions of E1A in cardiac myocytes.

4/9/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

09922281 BIOSIS NO.: 199598377199
Involvement of the cell-cycle inhibitor Cip1/WAF1 and the E1A-associated p300 protein in terminal differentiation.
AUTHOR: Missero Caterina(a); Calautti Enzo(a); Eckner Richard; Chin Jeannie (a); Tsai Li Huei; Livingston David M; Dotto G Paolo(a)
AUTHOR ADDRESS: (a)Cutaneous Biol. Res. Cent., Massachusetts General Hosp., Charlestown, MA 02129**USA
JOURNAL: Proceedings of the National Academy of Sciences of the United States of America 92 (12):p5451-5455 1995
ISSN: 0027-8424
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The mechanism of cell cycle withdrawal during terminal differentiation is poorly understood. We report here that the cyclin-dependent kinase (CDK) inhibitor p21-Cip1/WAF1 is induced at early

times of both keratinocyte and myoblast differentiation. p21-Cip1/WAF1 induction is accompanied by a drastic inhibition of total Cdk2, as well as p21-Cip1/WAF1-associated CDK kinase activities. p21-Cip1/WAF1 has been implicated in p53-mediated G-1 arrest and **apoptosis** . In keratinocyte differentiation, Cip1/WAF1 induction is observed even in cells derived from p53-null mice. Similarly, keratinocyte differentiation is associated with induction of Cip1/WAF1 **promoter** activity in both wild-type and p53-negative keratinocytes. Induction of the Cip1/WAF1 **promoter** upon differentiation is abolished by expression of an adenovirus E1A oncoprotein (dl922/947), which is unable to bind p105-Rb, p107, or cyclin A but which still binds the nuclear phosphoprotein **p300** . Overexpression of **p300** can suppress the E1A effect, independent of its direct binding to E1A. Thus, terminal differentiation-induced growth arrest in both keratinocyte and myoblast systems is associated with induction of Cip1/WAF1 expression. During keratinocyte differentiation, Cip1/WAF1 induction does not require p53 but depends on the transcriptional modulator **p300** .

4/9/6 (Item 6 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

09825687 BIOSIS NO.: 199598280605

Adenovirus E1A represses cardiac gene transcription and reactivates DNA synthesis in ventricular myocytes, via alternative pocket protein- and p300 -binding domains.

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JOURNAL: Journal of Biological Chemistry 270 (14):p7791-7794 1995
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: To examine the potential impact of disrupting "pocket" protein function on cardiac differentiation and growth, we introduced 12 S E1A genes into neonatal ventricular myocytes, by adenoviral gene transfer. In the absence of E1B, E1A was cytotoxic, with features typical of **apoptosis** . In the presence of E1B, E1A preferentially inhibited transcription of cardiac-restricted alpha-actin **promoters** , and reactivated DNA synthesis in cardiac myocytes, without cell death. Mutations that abrogate known activities of the amino terminus of E1A, versus conserved region 2, demonstrate that the "pocket" protein- and **p300** -binding domains each suffice, in the absence of the other, for transcriptional repression and re-entry into S phase.

4/9/8 (Item 2 from file: 34)

DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

03905301 Genuine Article#: QR526 Number of References: 39

Title: ADENOVIRUS E1A REPRESSES CARDIAC GENE-TRANSCRIPTION AND REACTIVATES DNA-SYNTHESIS IN VENTRICULAR MYOCYTES, VIA ALTERNATIVE POCKET PROTEIN-BINDING AND P300 **-BINDING DOMAINS**

Author(s): KIRSHENBAUM LA; SCHNEIDER MD
Corporate Source: BAYLOR COLL MED,DEPT MED,MOLEC CARDIOL UNIT,1 BAYLOR PLAZA,RM 506C/HOUSTON//TX/77030; BAYLOR COLL MED,DEPT MED,MOLEC CARDIOL UNIT/HOUSTON//TX/77030; BAYLOR COLL MED,DEPT CELL BIOL/HOUSTON//TX/77030; BAYLOR COLL MED,DEPT PHYSIOL & MOLEC BIOPHYS/HOUSTON//TX/77030

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Journal Subject Category: BIOCHEMISTRY & MOLECULAR BIOLOGY

Abstract: To examine the potential impact of disrupting ''pocket'' protein function on cardiac differentiation and growth, we introduced 12 S E1A genes into neonatal ventricular myocytes, by adenoviral gene transfer.

In the absence of E1B, E1A was cytotoxic, with features typical of **apoptosis** . In the presence of E1B, E1A preferentially inhibited transcription of cardiac-restricted alpha-actin **promot rs** , and reactivated DNA synthesis in cardiac myocytes, without cell death. Mutations that abrogate known activities of the amino terminus of E1A, versus conserved region 2, demonstrate that the ''pocket'' protein- and **p300** -binding domains each suffice, in the absence of the other, for transcriptional repression and re-entry into S phase.